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MAMMALIAN ADAPTATIONS TO DIVING
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by

Michael B. Strauss
Lieutenant, MC, U.S. Naval Reserve

Bureau of Medicine and Surgery, Navy Department
Research Work Unit MR011.01-5013.01

Released by:

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COMMANDING OFFICER
Naval Submarine Medical Center

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SUMMARY PAGE

THE PROBLEM

To prepare a comprehensive review of the literature dealing with mammalian adaptations to diving, with particular emphasis on respiratory and cardiovascular changes.

FINDINGS

This review suggests that there is a remarkable integration of the adaptations and acclimatizations involved in the breathhold diving response. A new pathophysiological mechanism for understanding the etiology of decompression sickness is presented, and applications of responses associated with diving to clinical medicine and the prediction of diving performance are dicussed.

APPLICATIONS

This report presents a body of material that will be valuable for all submarine medical officers, students in the School of Submarine Medicine, and individuals interested in research into human adaptation to underwater situations.

ADMINISTRATIVE INFORMATION

This study was performed and reported as a part of the requirements for qualification as a Submarine Medical Officer, and the thesis was selected for publication in order to make the material available to the School of Submarine Medicine, to the Technical Library, SubMedCen, and to medical officers in the Submarine Force. It has been designated as SubMedResLab Report No. 562, and Report No. 1 on In-House Research Work Unit, MR011.01-5013. It was approved for publication on 28 January 1969.

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ABSTRACT

This thesis is a comprehensive review of the literature dealing with mammalian adaptations to diving. Respiratory and cardiovascular changes are particularly emphasized. Other subjects pertaining to diving adaptations, such as fluid conservation, temperature maintenance, propulsion, underwater vision, and navigation are also included in the discussion.

This review suggests that there is a remarkable integration of the adaptations and acclimatizations involved in the breath-hold diving response. On the basis of these studies, a new pathophysiological mechanism for understanding the etiology of decompression sickness is presented in the conclusion. In addition, applications of responses associated with diving to clinical medicine and prediction of diving performance are discussed. Wherever possible, comparison of human breath-hold divers to aquatic mammals is made.

MAMMALIAN ADAPTATIONS TO DIVING

INTRODUCTION

From the beginning of recorded history man, has attempted to extend the confines of his terrestrial environment. Greek mythology is replete with references to this. One need only think of the personification of Icarus, Zeus, Hermes, and Poseidon. Records of human diving experiences are noted several millennia ago in Homer's Iliad and Herodotus' writings.[30] Since Aristotle's time, it was recognized that the porpoise was a mammal and could tolerate the aquatic environment much better than its terrestrial counterparts. However, no systematic investigation of this subject was made until Paul Bert's study of diving vertebrates in the 1870's. Approximately seventy years later, Scholander and Irving's classical studies delineated many of the physiological changes that account for the extraordinary diving feats of seals and other aquatic mammals. Subsequently, it was recognized that humans, to a limited extent, could adapt to the aquatic environment. What is the scope of mammalian adaptations to the aquatic environment? Table I is a comparison of maximum breathhold times and depth excursions in a variety of mammals. It is apparent that there is a continuum of responses beginning with the non-diving mammals, progressing to the conditioned human diver, and ending with the aquatic mammals. These diving abilities are a manifestation of the absence or presence and degree of development of the physiological adaptations which make breathhold diving possible. The most profound and generalized changes are observed in the cardiovascular and respiratory systems. Also, there are important adaptations in regard to temperature maintenance, water conservation, propulsion, and other special problems peculiar to the aquatic environment. This thesis will discuss these adaptive changes and their underlying physiological mechanisms. Special emphasis will be given to the presence and absence of these adaptations in the human breathhold diver.

A. RESPIRATORY AND CARDIOVASCULAR ADAPTATIONS

Aquatic mammals demonstrate multiple adaptive changes in their respiratory systems. Several of these changes are also observed in the conditioned human diver. The first question that may be asked is, how important are the lungs as an oxygen reservoir during diving? Repeated observations reveal that seals, whales and porpoises dive after complete exhalation. This is in direct contrast to the human breathhold diver. Table II is a comparison of breathhold times based on pulmonary oxygen stores during resting conditions and in conjunction with the oxygen conserving (diving) reflex. When these figures are examined and the predicted and observed breathhold times are compared, it is apparent that the lungs, at best, would satisfy only one-third to one-half of the aquatic mammal's oxygen requirements during the dive. Since the lungs do not function as an oxygen storage reservoir in the aquatic mammal, what respiratory adaptations have these animals made? Four changes can be observed. First, there are adaptations to resist the effects of thoracic squeeze. Second, there are changes which eliminate the possibility of developing decompression sickness. Third, there are adaptations which make the organism less responsive to the effects of hypoxia and hypercapnia. Finally, there are those changes which improve the efficiency of ventilation during the surface interval and recovery phase of the breathhold dive.

Before these adaptations are considered, it is appropriate to discuss the effects that increased ambient pressure has upon the respiratory system. As the ambient pressure is increased, the lung volume is compressed in accordance with Boyle's Law. At some pressure, the lung volume would be compressed to a point where either tissue fluid would pass into the alveolar sacs or the chest wall would collapse in order to counteract the differential in pressure. When this theoretical limit is exceeded, the condition termed thoracic squeeze

TABLE I: BREATH-HOLD TIMES AND UNASSISTED DEPTH EXCURSIONS IN MAMMALS

Animal	Breath-hold Time (Minutes)	Maximum Recorded Depth (Feet)	References
Beaver	15	Not Available	6, 55, 65
Cat	3	N.A.	4, 6
Dog	4, 4½	N.A.	4, 6, 65
Muskrat	12	N.A.	6, 65
Rabbit	3	N.A.	6, 65
White Rat	2	N.A.	6, 65
Blue Whale	50	N.A.	6, 65
Bottle Nose	120	2700	6, 65, 121
Fin Whale	30	N.A.	6, 65
Sperm Whale	75	3300	6, 65, 83
Grey Seal	18, 20	440	2, 6, 55
Harbor Seal	23	825	83
Sea Lion	30	450, 550	55, 83
Weddell Seal	43	1800	83
Human	½, 6*, 15**	212½*	29, 55, 65
Killer Whale	12	N.A.	137
Porpoise	6	1000	68, 140
Manatee	30	N.A.	129

*Personal experience (Cf. Reference 29)

**After breathing 100% oxygen

TABLE II: ESTIMATED DIVE DURATION BASED ON PULMONARY OXYGEN STORES

Animal	O_2 Available (ml)	Relative Lung Volume (ml/100 kg)	Resting O_2 Consumption (ml/min)	Predicted Dive Time (min)	O_2 Consumption Dive Reflex (ml/min)	Observed Dive Time (min)	References
Seal	1520	5.0	250	6	84	18	4, 6
Bottlenose	109×10^3	2.5	3500	36	900	120	4, 6, 65
Fin Whale	3350×10^3	2.9	200×10^3	17	110×10^3	30	4, 6, 65
Human	900	7.0	400	2¼	Not Available	Cf. Table I	65, 129
Porpoise	1090	6.9	450	2½	N.A.	6	4

occurs. In diving there are pronounced changes in the diffusing capacity of the human lung. Guyatt, et al., found that mere immersion to the level of the neck increased the diffusing capacity of the lungs by sixteen percent.[51] These results were thought to be due to pulmonary vascular engorgement, secondary to the hydrostatic pressure gradient which forces blood from the large venous reservoirs in the legs to the central circulation. Craig and Ware reported that the vital capacity was reduced significantly by immersion to the neck level.[28] Residual lung volume was also reduced, but to a lesser degree. As the ambient pressure increases in breathhold dives to depths greater than fifty feet, pulmonary-arterial (pulmonary vein) oxygen extraction improves and pulmonary-venous (pulmonary artery) carbon dioxide gradients reverse.[120] This has two important effects on the breathhold dive performance. First, at depth it reduces the desire to breathe and tends to increase the breathhold time. Second, on ascending, these gradients tend to return to normal proportions. The sudden elevation in pCO_2 and the reduction in pO_2 develops an extreme desire to breathe. Hypoxia and syncope are possible sequelae, especially in the overzealous breathhold diver. Decrease of pO_2's to thirty-five millimeters of mercury have been recorded during the ascent.[120] It is unlikely that these effects interfere with the diving ability of the aquatic mammal, as will be explained shortly.

The ability of the aquatic mammal to resist thoracic squeeze is important, especially when one considers the extreme depths to which these animals dive. It is interesting to note that in 1823, S. N. Smith stated, "... if the depth be considerable, the water on the breast and organs is so great that it occasions the eyes to become bloodshot and produces spitting of blood, and if the practice is persisted in, it will most likely prove fatal."[30] This well describes the thoracic squeeze syndrome. Theoretically, this condition would appear when the pressure differential was great enough to compress the total lung volume to an amount smaller than the residual lung volume. The pathophysiology of these effects was described previously. In the average sized human, after a maximum inspiratory effort, lung damage would occur with pressurization to approximately four atmospheres, equivalent to a depth of one hundred feet. Diving mammals compensate for this tremendous pressure differential in three ways. First, their thorax is much more elastic than that of the human.[2] This flexibility would ameliorate, to some extent, the intrathoracic volume changes necessary to produce thoracic squeeze. Second, the lungs of seals and whales can collapse to a point where they become atelectatic and yet not separate from the chest wall.[2] During the dive, total lung capacity would approach the dead space volume. Consequently, the alveolocapillary exchange of gas would be zero, again suggesting that gas stores in the lungs are not important in the aquatic mammal. Third, venous blood may compensate for the space that develops when the lungs collapse. Demonstrations in seals of large venous sinuses adjacent to the inferior vena cava and a sphincter effect by the diaphragm support this hypothesis. The inferior vena caval diaphragmatic sphincter mechanism may also be important in preventing overloading the heart with increases in ambient pressure.[55] Since blood is a liquid and is non-compressible, extreme depth excursions without thoracic complications become possible.

In regard to human divers, these adaptations are virtually non-existent. The one important change which increases the theoretical depth to which a human breathhold diver can descend safely is a significant increase in the total lung capacity and a relative decrease in the residual volume of the lung.[124]

These adaptive changes to increased pressure lead directly into the discussion of decompression sickness in the diving mammal. This phenomenon was not thought to occur in the breathhold diver. In the manual, Submarine Medicine Practice, it is stated that "decompression sickness is virtually impossible for the skin diver because he cannot submerge deep enough or remain long enough to take up a troublesome amount of nitrogen —unless he has access to a supply of air at depth."[138] However, Paulev has demonstrated that this is incorrect.[100] In four divers who

participated in a series of repetitive breathhold dives, symptoms of decompression sickness developed. The divers descended to 65 feet and had bottom times approximating 2 minutes. Surface intervals varied from a few seconds to a maximum of two minutes. After five hours of diving, which included about 60 dives, symptoms and signs of decompression sickness were noted while surfaced. The diagnosis was substantiated by complete remission of the symptoms with recompression. Subsequently, Paulev measured the accumulated tissue nitrogen tensions in repeated breathhold dives and found that it was possible to exceed the maximum tensions permitted for no decompression dives.[102] These studies provide a possible physiological explanation for the Taravana Syndrome described by Cross in the Polynesian divers of Tuamotu.[30] These divers demonstrate predominantly central nervous system manifestations of what appears to be a decompression sickness. As in Paulev's experiences, symptoms were noted after an exceedingly vigorous 4 to 5 hour dive session which often included descents to 165 feet and very short surface intervals. This problem is not observed on an adjacent island where the divers descend to equivalent depths, but have surface intervals two to three times the duration of the Tuamotu diver. Unfortunately, no decompression facilities were available to treat the affected individuals and positively verify that the symptoms were entirely due to decompression sickness.

Aquatic mammals have made a number of adaptations to eliminate the occurrence of decompression sickness in their extraordinary diving feats. First, as noted before, these animals dive after full exhalation. The amount of nitrogen available to saturate the tissues is reduced by 80 to 90%. Explanations for diving after exhalation might be related to facilitation of the diving bradycardia and peripheral vasoconstriction responses, rather than specifically for prevention of decompression sickness. This may also help achieve a negative buoyancy to assist in descent. It is interesting to note that the Ama of Japan do not dive after maximal inspiration either. Decompression sickness has not been documented in these divers. The Ama descend after inhaling to 85% of their total lung capacity.[57] Further inhalation may cause discomfort and make descent more difficult. What is the effect of diving after full exhalation? By the time a depth of 100 feet is reached, virtually all alveolar nitrogen has passed into the tissues. Further submersion would not result in additional nitrogen deposition into the tissues. The second factor helpful in preventing decompression sickness is related to the adaptation previously observed to avoid a lung squeeze. If the lung becomes atelectatic, then the small residual volume of air would be forced into noncollapsible, nondiffusable sections of the respiratory system, namely the bronchi and trachea. Thus, further dissolution of nitrogen into the body tissues would not be possible.

The third respiratory adaptation relates to observations that aquatic mammals are less sensitive to low alveolar oxygen concentrations and high carbon dioxide levels than nonaquatic mammals. In the seal, the resting P_aCO_2 is higher and the P_aO_2 is lower than those in the human. Aquatic mammals are known to have a depressed ventilatory response to elevated carbon dioxide and reduced oxygen tensions. This may be an adaptive mechanism to increase the duration of the dive. However, these mammals are not insensitive to the alveolar carbon dioxide level. Andersen reported that elevated carbon dioxide tensions did increase the ventilatory response in the seal, but not as much as the response in the human subject.[4] This corresponds to Irving's observations that ten percent concentrations of carbon dioxide cause only small respiratory increments in the beaver and muskrat.[63] Analogous changes can be observed in the trained human diver. Schaefer reported that submarine escape training tank instructors at the United States Naval Submarine Base, New London, Connecticut, demonstrated a number of respiratory adaptations when compared to a control group of nondiving laboratory personnel.[122] The instructors were observed to have significantly decreased ventilatory responses to 10½% carbon dioxide, better oxygen utilization, acceptance of larger ox-

ygen debt, and increased tolerance to elevated tissue carbon dioxide levels. These responses disappeared three months after diving was discontinued. This suggests that physical conditioning is necessary for development and maintenance of these adjustments. Markedly elevated carbon dioxide and tidal volumes are observed in trained underwater swimmers.[46] Similar findings have been reported in the Ama of Japan.[35] These adaptations are noteworthy for they are the first of those discussed where analogous responses can be seen in the aquatic mammal, and second, they reveal that practice can improve the breathhold diving performance.

Finally, there are those respiratory adaptations which improve the efficiency of air exchange and reduce the recovery time after a stressful dive. Table III is a comparison of the relative respiratory functions of aquatic mammals and humans. The data suggest that man's respiratory exchange function is relatively inefficient when compared to these animals. Although relative lung capacities are similar in man, seal, and porpoise, these latter two have markedly slower respiratory rates, much greater oxygen utilization percentages and larger tidal volumes. Whales, although much larger, reflect similar changes. The large tidal volume/total lung capacity ratio results in a large exchange of gas during each respiratory cycle and compensates for the low respiratory rate. The low respiratory rate also makes greater utilization of oxygen possible, as this table indicates. Andersen observed that as respiratory rate increases, the tidal volume decreases and pulmonary ventilation is reduced.[4] The effect of these changes is fourfold. First, the low respiratory rate and large volume of air exchanged with each cycle reduce the energy expenditure required for ventilation. Second, the elevated oxygen utilization increases the efficiency of each breath and eliminates the need to increase the respiratory rate in order to maintain the same level of ventilation. Third, the fully inflated lung may assist in buoyancy. Active swimming movements would then be minimized and energy expenditures correspondingly reduced. Porpoises spend intervals on the surface in a semiconscious state. This may be analogous to sleep.[137] During this period the porpoise is apneaic. When a breath is required, the animal rolls to his side, makes several apparently reflexive movements of his pectoral fins in order to clear the blow hole above the water surface, exhales, quickly inspires, and then resumes his former attitude. Maintenance of the lung volume may be important in achieving a positive buoyancy in this situation. Also, these reflexive movements may be important in preventing drowning, for it is known that porpoises are very sensitive to interference with breathing. Should water enter the blow hole during respiration, the porpoise becomes apneaic and asphyxiates without struggling to breathe.[68]

Improved efficiency in respiratory exchange can be demonstrated in the well-trained human breathhold diver. Schaefer has reported significant increases in inspiratory reserve volumes, tidal volumes, vital

TABLE III: RELATIVE RESPIRATORY FUNCTIONS IN DIVING MAMMALS

Animal	Resp. Per Min.	Relative Ventilation (L/min/100 kg)	Relative Lung Cap. (L/100 kg)	Relative Tidal Vol. (L/100 kg)	Tidal Vol. / Lung Cap. Ratio	Oxygen Utiliz. (%)	References
Bottlenose	1-2	3	2.5	2.2	0.88	8-10	4, 129
Fin Whale	1-2	3	2.9	2.5	0.86	8-10	4, 129
Human	15	12	5.0	0.8	0.16	4- 5	4, 129
Manatee	1	2	5.1	2.9	0.57	7-10	4, 129
Porpoise (Phocaena)	2-4	5	6.9	5.4	0.78	8-10	4, 68, 129
Porpoise (Tursiops)	1	6	6.6	5.9	0.89	8-10	4, 68, 129
Seal	3-4	14	5.0	1.8	0.36	5- 7	4, 129

capacities, and total lung capacities after repeated breathhold diving.[124] Quantitatively, vital capacities were over fourteen percent higher in escape training tank instructors than predicted.[17] The residual volumes decreased relatively. These changes result in increased respiratory efficiency and an improved ability to resist thoracic squeeze. Schaefer also noted that there were quantitatively different responses to breathing elevated levels of carbon dioxide.[123] High and low ventilatory response groups were defined. The low ventilatory response group uniformly had larger tidal volumes, lower respiratory rates, and improved tolerance to elevated alveolar carbon dioxide tensions than the high ventilatory group. These observations are consistent with the reported changes in vital capacity and total lung capacity of the Ama.[135] The final effect of this improved efficiency in respiration relates to conservation of body heat and will be discussed in the next section of this paper.

The cardiovascular adaptations observed in the aquatic mammal are even more remarkable than the respiratory changes. In contrast to the respiratory adaptations which tend to be instrumental in protecting the aquatic mammal from medical problems associated with diving, the cardiovascular changes are responsible for improving the duration of the dive. To some extent, the cardiovascular adaptations reflect a general response to anoxia and can be demonstrated in many mammals.

The bradycardia reflex is the most well-documented of these responses. Table IV summarizes this response in a variety of mammals. Marked slowing of the heart has been observed in all aquatic mammals, including man, with immersion, and in many terrestrial mammals when subjected to anoxic stresses. The most profound changes are seen in the seal, where heart rates have been observed to slow to ten percent of their resting level on commencement of diving.[67] In the porpoise, the heart slows to about fifty percent of resting levels, and, in contrast to the seal, the bradycardia is of gradual onset.[38] The immersion bradycardia observed in the human very much resembles that observed in the porpoise. The onset is usually gradual, and after thirty seconds the rate slows to fifty percent of normal.[58] Although all investigations did not give unanimous conclusions, it appears that immersion in water does slow the heart in the human regardless of the individual's sex, swimming ability, or familiarity with the water, provided the immersion did not provoke an anxiety reaction. Immersion bradycardia has been observed in children. Craig reported that this response was independent of depth to dives of eighty feet.[24]

TABLE IV: BRADYCARDIA RESPONSE TO IMMERSION

Animal	Mode of Onset	Resting Pulse	Pulse Rate Immersion Bradycardia	Pulse $\frac{\text{Immersion}}{\text{Pulse Rest}}$	References
Beaver	Gradual	75-90	0-10	0.0-0.11	66
Hippopotamus	Gradual	100	10-20	0.10-0.20	34
Platypus	Slow	140	20	0.14	73
Human	Gradual	75	40-50	0.53-0.67	126
Killer Whale	Gradual	60	30	0.50	137
Porpoise	Gradual	60	30	0.50	38
Seal	Rapid	70-140	7-14	0.10	36, 50
Whale	N.A.	100	12-24	0.12-0.24	82

Before reviewing the proposed mechanism for the bradycardia reflex, it is important to investigate some of the influencing factors. First of all, in humans, the effect is much more pronounced if the subject is immersed in water than if breath holding is performed in the air.[60] As in aquatic mammals, this response is not obliterated by exercise.[119] Bradycardia is not reproducible by pressurization in the hyperbaric chamber.[24] Temperature has an important effect. A twenty percent greater bradycardia effect was observed during the winter than in the summer in the Ama of Japan.[60] This temperature effect has been reproduced in the laboratory with facial wetting. In fact, Whayne says, "Thus, there can be no doubt that the colder the facial stimulus, the more intense the depression of heart rate and rhythm."[147] Snorkel breathing while immersed was found to abolish the bradycardia.[53] Finally, the mere placement of a wet towel over the subject's face causes a heart response analogous to immersion in water.[15, 53, 147]

Several hypotheses have been proposed to explain the underlying physiological mechanisms for these observations. First, it is known that increasing the venous return to the heart will slow the pulse. This may be initiated by a number of mechanisms. Tilting the head downward, vertical immersion in water, elevation of the feet, and the valsalva maneuver have been reported to slow the heart.[24] Harding, et al., suggest that the bradycardia is reflexly initiated by distention of stretch receptors in the atrial walls. This occurs secondary to increased volume of blood in the thorax, resulting from the hydrostatic effect of the immersion.[53] In contrast, if the venous return is compromised, the opposite effect, that is to say, a tachycardia is observed. This may be secondary to a reduction in blood pressure. While this concept may be valid for a rather restricted situation in humans where breath holding is accompanied by large inspiratory volumes and changes in intravascular fluid distribution, it is insufficient to account for the bradycardia reflex observed in the aquatic mammals.

A reflex initiated by stimulation of the trigeminal nerve has also been hypothesized to explain the diving bradycardia response.[5] This mechanism is consistent with the rapid initiation of the response, its independence from anoxia and pressure, and the importance of facial immersion. The afferent end organ is in the face. Andersen identified the ophthalmic branch of the trigeminal nerve as the afferent nerve pathway in the duck.[5] This explains why an aquatic mammal will asphyxiate in about one third the time, when its trachea is occluded and the animal is left to die in the air, than it would if the procedure is repeated in the water.[149] The bradycardia response in the seal has been initiated by manually closing its nostrils.[55] The efferent side of the reflex is mediated through the vagus nerve. Atropine which is a parasympatholytic agent abolishes the diving bradycardia and thus lends further support to the hypothesis that this response is mediated through the nervous system.[96, 98]

In the human, and perhaps the aquatic mammal, definite supratentorial influences are apparent. The syncope associated with venopuncture and the observation that emotionally significant situations were capable of inhibiting and facilitating this response, suggest that, in the human, it is modified by the higher centers in the brain.[149] Irving, et al., have observed analogous situations in the seal.[70] They found that seals with ". . . steady dispositions and apparently stable mental backgrounds proved invariably to be the better divers under experimental conditions." [70] Murdaugh, Seabury, and Mitchell reported that anticipation of a dive, developed by operant learning techniques, did not decrease the heart rate in seals while ascent gradually abolished the bradycardia response.[96] Harrison and Tomlinson observed increases in seals' heart rates as the dive progressed.[55] This may reflect myocardial ischemia and act as an automatic signal to surface. However, if the seal approached the surface and decided to descend again before surfacing, the bradycardia returned.[96]

Although electrocardiograms of aquatic mammals while diving indicate that the bradycardia is well-tolerated, this is not universally true with the well-trained human

diver. Arrhythmias are the most common electrocardiographic abnormality in the human breathhold diver. With the exception of occasional paroxysmal ventricular contractions, the arrhythmias are primarily inhibitory in type.[99] Atrioventricular blocks and peaked "T" waves occur. These effects are exaggerated by immersion in cold water. Seals who have their airways occluded demonstrated similar electrocardiographic changes.[95] In a study of human divers by Olsen, et al., arrhythmias were observed in over 75% of the subjects.[99] Cessation of the arrhythmias occurred promptly after surfacing. The electrocardiogram of the diver, both human and non-human, is characterized by prolonged diastole, increased "QT" intervals and progressively increased "PR" intervals to a point where the "P" wave is lost. Conditioning in the human diver does not appear to have any effect on the electrocardiogram. Perhaps this indicates that the human breathhold diver does not belong below the surface of the water.

It is obvious that bradycardia alone cannot account for the long dive durations observed in aquatic mammals. Unless there were compensatory vascular, hematological, and metabolic changes, bradycardia, in itself, would be detrimental to the organism.

The first of these compensating factors is a profound peripheral vasoconstriction and a preferential shunting of blood from the extremities to the great vessels, heart, lungs, and brain. This permits the oxygen stores in the blood to be used almost exclusively to perfuse the heart and the brain. The bradycardia markedly lowers the heart's oxygen requirements. The brain would be expected to maintain its usual oxygen utilization rate during the dive. To illustrate this, an example of oxygen requirements in the seal is used. Harbor seals have a resting oxygen consumption rate of three hundred milliliters per minute. Of this, forty to fifty milliliters per minute are required to perfuse the brain. The blood oxygen stores of the seal are about one thousand milliliters. If no compensating mechanisms were utilized, the maximum predicted dive duration would be slightly over three minutes. After the oxygen conserving reflex is initiated and peripheral vasoconstriction is in effect, the oxygen dissolved in the blood would theoretically be capable of supplying the brain's oxygen requirements for twenty to twenty-five minutes which corresponds closely with the maximum observed dive times in the seal.[16, 71] Table II also compares the predicted and observed breathhold dive times based on pulmonary oxygen stores in man and four aquatic mammals. It indicates that the actual diving times, except for the human, are two to three times those predicted and suggests this oxygen conservation mechanism is relatively uniform in the diving mammals studied.

The compensating factor of peripheral vasoconstriction has also been well documented. It is so profound in the diving mammals that blood almost ceases to flow in these areas. Incisions through muscles and skin do not bleed while the aquatic animal is submerged.[104] Immediately upon surfacing, profuse bleeding occurs. Temperatures in the extremities decreased during dives to a degree comparable with tourniquet application to these areas.[128] Murdaugh, et al., reported that, ". . . the degree of arterial constriction, as indicated by the dye-injection studies, is so extreme during diving that it is as if these areas were surgically removed from the animal."[94] During the dive, renal blood flow ceases and the seal becomes anuric.[94] Atropine does not block the renal ischemia response induced by breathhold diving in the aquatic mammal.[13] Perhaps this response is independent of the vagal reflex associated with the bradycardia. Arterial vasoconstriction also occurs in the vasculature of the skin, splanchnic beds, and extremities. In the seal, iliac blood flow ceased immediately after submerging.[144] It even preceded the bradycardia. Struggling caused only a slight increase in iliac blood flow. However, Irving, Scholander, and Grinnell reported that the blood pressure in the submerged seal is maintained at a normal level in the femoral artery, and state that vasoconstriction more peripherally is the prime factor in this vascular response. These same adaptations, only to a lesser degree, have been observed in the human subject. Elsner and Scholander noted

that limb blood flow, as measured by occlusion plethysmography, fell "nearly to zero" during breathhold dives in humans.[39] The effect was much more profound with facial immersion in water than with simple breathholding in air. This provides additional evidence that the peripheral vasoconstriction response is related to the bradycardia and is of reflex origin. Vasoconstriction may be mediated in conjunction with and by the same mechanism as the bradycardia response. Van Citters, et al., found that in the elephant seal electrical stimulation of the periventricular area of the anterior hypothalamus produced a bradycardia and peripheral vasoconstriction similar to that observed during the breathhold dive.[144]

The absence of peripheral blood flow, apparent during the shock syndrome, and the bradycardia associated with the fetal distress syndrome indicate that these changes represent a generalized oxygen conservation mechanism, rather than a specific adaptation to diving.

Continuous perfusion of the brain and heart, in spite of the profound bradycardia, is essential. Without this compensation, bradycardia, in itself, would be of no value to the aquatic mammal. Intra-arterial blood pressure studies performed on seals reveal that mean central blood pressure is maintained at approximately normal levels. The systolic pressure is unchanged from the eupneaic period. The important compensation is in the diastolic blood pressure which reveals a gradual and prolonged decrease until the next systole.[128] This insures continuous perfusion of the brain between the systoles. Several morphological changes in the aquatic mammal's vascular system make this possible. First, the energy for maintaining the diastolic blood pressure is a result of the stretching of the highly elastic blood vessel walls during and immediately after systole. Second, an elastic, bulbous enlargement of the aorta, just distal to the left ventricle, is present.[37] This almost doubles the diameter of the aorta. This structural change has been observed in the seal, porpoise, and whale. In effect, "this aneurysm" may act as a passive heart and help to insure perfusion of the brain during the prolonged diastolic period. There is a transient reversal of blood flow in the abdominal aorta after each heart beat. This oscillatory phenomenon reflects the highly elastic nature of the blood vessel walls and indicates that blood is being pumped against a tightly constricted arterial tree. Again, these changes help to maintain the central blood pressure and provide adequate perfusion of the brain during the astyolic period.

The increased ability for the noncritical tissues in the aquatic mammal to function anaerobically and remain isolated until the dive is complete is the third compensating adaptation associated with the diving bradycardia and peripheral vasoconstriction responses. Muscle tissue has the ability to function anaerobically in the absence of oxygen. Energy production, however, is only a fraction of what could be produced aerobically from the same quantity of metabolites. Yet, it is enough to sustain the noncritical tissues during the dive. As the anaerobic metabolic processes continue, an oxygen "debt" is incurred. This must be corrected during the recovery period. The production of lactic acid is the hallmark of anaerobic metabolism. Studies in the seal reveal that several minutes after submergence, oxygen disappears from its muscle tissue, although the arterial blood is still well saturated with oxygen.[125] When muscle oxygen stores are exhausted, lactic acid rapidly accumulates in the muscle tissue. Very little lactic acid escapes into the central circulation until breathing is resumed. In addition, the well-oxygenated blood in the central vasculature does not transfer its oxygen to the muscle hemoglobin. This, again reflects the effectiveness of the vasoconstriction response in shunting blood from the periphery. With severe anoxia, this circulation barrier ultimately breaks down. Activity increases the amount of oxygen "debt" and the rate of lactic acid formation. Studies of Scholander, et al., indicate that metabolism decreases exponentially as the dive progresses as a compensatory mechanism.[128] Schaefer has demonstrated analogous responses in the well-conditioned human breathhold diver.[121] Dur-

ing the breathhold dives of instructors at the Submarine Escape Training Tank, New London, Connecticut, metabolic rates were observed to decrease during dives. Schaefer reported that the lactic acid contents of the divers' blood rose from an average pre-dive level of nine milligrams percent to 55 milligrams percent one minute after a dive to 90 feet was completed.[121] Five minutes after surfacing, the serum lactic acid had decreased to 25 milligrams percent. During the dive period, an oxygen "debt" of 1,400 milliliters and a carbon dioxide excess of 900 milliliters accrued.[124] Scholander reported similar findings in the breathhold divers of the Thursday Islands.[126] Analogous findings were observed in the manatee, where serum lactic acids of 10 milligrams percent occurred during the dive and levels varying from 100 to 150 milligrams percent appeared after surfacing.[129] These changes were accompanied by a marked rise in the respiratory quotient.

The fourth adjustment which complements the bradycardia and peripheral vasoconstrictive response is the increased ability to carry, store, and utilize oxygen by the aquatic mammal. Increased blood volume, elevated hematocrits, and more extensive stores of myoglobin are the three factors which improve the oxygen-carrying and storage abilities of the blood of aquatic mammals. Irving, et al., state that diving mammals have a fifty percent greater oxygen storage capacity than terrestrial mammals of corresponding size.[71] Blood volumes, expressed as percentage of total body weight, are approximately two times as great in the aquatic mammals as in the nondiving mammal.[6] Diminution in the size of the red blood cell and increased red blood cell hematocrits also contribute to this oxygen storage. Hematocrit concentrations are in the range of fifty percent for the aquatic mammals studied. The third factor increasing the oxygen storage abilities of the aquatic mammal is the enriched stores of muscle hemoglobin which is frequently referred to as myoglobin. Myoglobin has a much higher affinity for oxygen than hemoglobin. Andersen observed that nearly fifty percent of the total amount of oxygen available during submersion is stored in the myoglobin.[6] It should be reemphasized that during the diving reflex, there is no transfer of oxygen from the low-ogygen affinity blood hemoglobin to the high-oxygen affinity muscle hemoglobin. Thus, the oxygen stores required to supply the brain and heart are not compromised. The oxygen stores in the myoglobin are usually exhausted after the first three or four minutes of the dive, and then energy production is limited to anaerobic metabolism in the muscle tissues.[6] Even though the myoglobin content is reduced to zero, the arterial blood is still fifty percent saturated at this time.[128] The fourth factor to be considered is the large venous reservoir observed in the seal and presumably present in the other diving mammals. The role of the sinuses and the diaphragmatic caval sphincter in preventing thoracic squeeze and fluid overload of the heart was alluded to previously. During the dive, these venous reservoirs contain approximately twenty percent of the total blood volume. Elsner, et al., observed that in the elephant seal, the arterial oxygen concentration fell more rapidly than the venous, and towards the latter half of the dive, the inferior vena cava did, in fact, have a greater concentration of oxygen than the arterial system.[41] These investigators suggested that the blood in the inferior vena caval system is a major oxygen storage depot during the dive. Finally, the decreased pH of the arterial blood, secondary to the lowered buffering power of the aquatic mammal's blood, increases the ability for the tissues to extract oxygen from the hemoglobin. In accordance with the Bohr formula, more oxygen is dissociated from oxyhemoglobin in the acid state than at the normal human serum pH. When all of these factors are combined, a fourfold increase in oxygen-carrying storage, and utilization is possible as Wasserman and MacKenzie have demonstrated in the seal.[146]

Two points are emphasized in summarizing the adaptive changes which improve the oxygen-carrying and storage capacities of acquatic mammals. First, the increased ability to store and utilize oxygen is no substitute for

the other factors associated with the diving response. Alone, these factors would account for only two or three minutes of submersion. However, when they accompany and complement the other mechanisms, they permit the extraordinary diving feats listed in Tables I and II. Second, increased hematocrits, decreased blood pH's and improved oxygen utilization have been demonstrated in the well-trained human breathhold diver.[121] These acclimitizations observed in the human are transient and, without continual diving, return to normal three months after diving is discontinued.

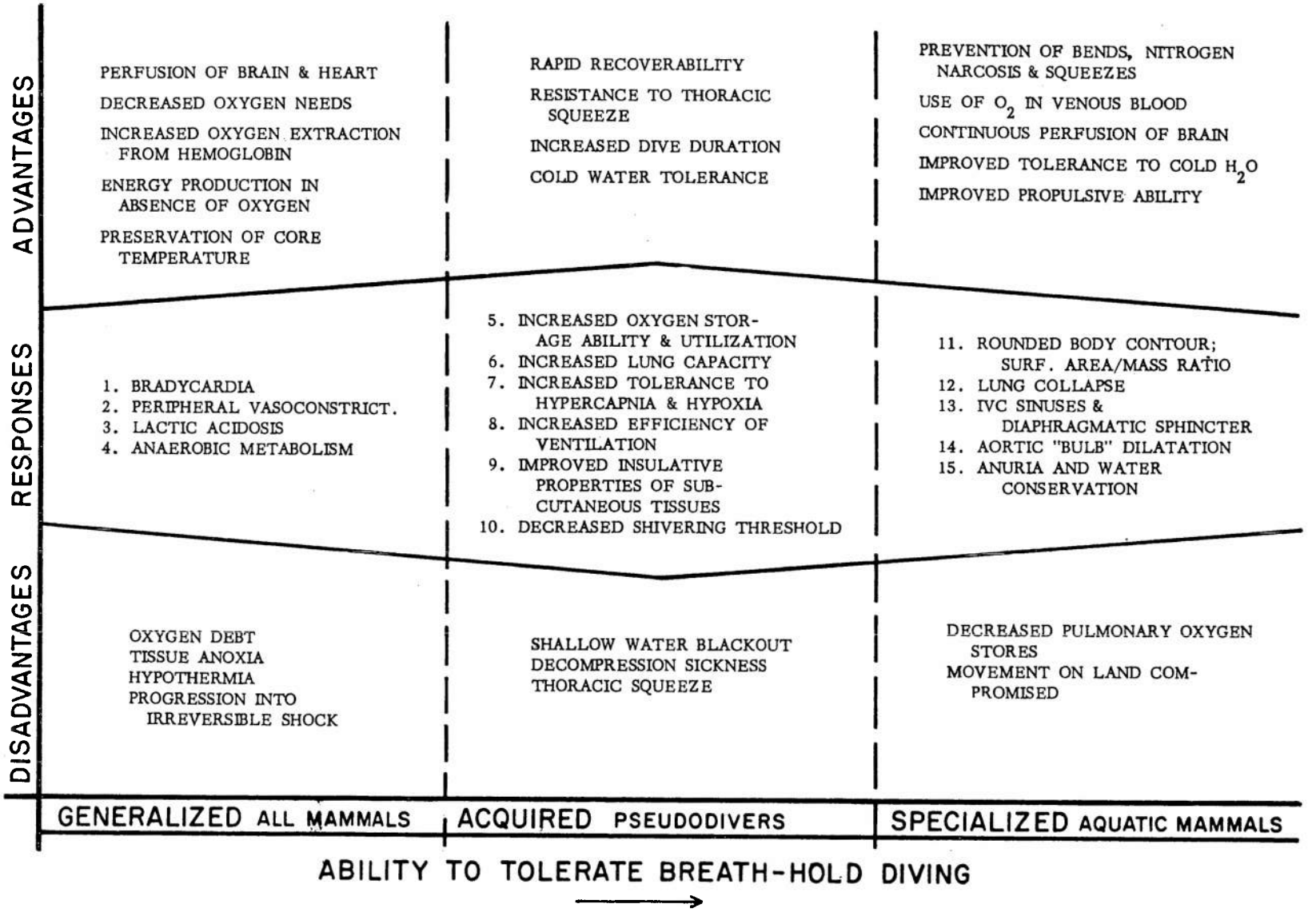
This concludes the discussion of the cardiovascular and respiratory adaptations. It should be noted that these changes can be divided into three main categories. First, there are those changes that are universal to the animal kingdom and occur spontaneously with anoxia. These include peripheral vasoconstriction, bradycardia, and decreased blood pH. As stated before, these three responses are often associated with the shock syndrome and hypothermia. The rapidity of onset, degree of change, and reversibility, in general, distinguish the aquatic mammals from the non-divers in regard to these responses to anoxia and immersion. The second group of cardiovascular and respiratory adaptations are those that are highly developed in the aquatic mammal, but can be developed in the non-diving mammal, especially man, with practice. These include the ability of the blood to transfer and carry more oxygen per unit volume, increased efficiency of ventilation, and improved ability to tolerate oxygen debts. Again, these changes are not confined to aquatic mammals and the "pseudo" diving mammal, but can be observed in the well-trained human athlete who participates in a high-repetition, low-resistance type of activity. The final group of changes are those peculiar to the aquatic mammal. These include the ability to collapse the lungs during submersion, the sequelae to this, namely the displacement of the lung volume with blood from venous reservoirs in the trunk, aneurysmal dilatation of the aorta, increased flexibility and compressibility of the thoracic wall, and diving after full exhalation. Undoubtedly, these adaptations are a means of preventing decompression sickness, thoracic squeeze, and nitrogen narcosis. They would be expected to decrease the oxygen storage capacities of the aquatic mammal, but other adaptations more than compensate for this effect. Chart A is a graphic illustration of the hierarchy of responses to apneaic diving, the advantages (in regard to breathhold diving) and disadvantages of each.

B. MISCELLANEOUS ADAPTATIONS TO DIVING

There are a number of difficulties the human encounters when diving which are not observed in the aquatic mammal. These include underwater visual acuity, ability to equalize the pressure between the middle and external ear, water conservation, tolerance to cold water exposure, and underwater navigation. Human divers demonstrate a wide range of individual variation in response to these problems. Practice and physical condition, in contrast to many of the respiratory and cardiovascular adaptations, have little effect in improving the human divers' ability to cope with these conditions.

Underwater vision is a problem to the human diver. The refractive index of water is four-thirds that of air. Thus, distances appear three-fourths as great in water as in air. Vision underwater is characterized by extreme hypermetrophia. The air-to-cornea interface, the primary image producing lens, is effectively obliterated by water which has nearly the same refractive index as the aqueous humor of the eye.[118] Thus, little refraction occurs at this interface. The human diver counteracts this effect by reestablishment of the air-to-cornea interface with the wearing of goggles or a mask. However, with descent, the pressure differential results in a negative pressure developing in the air cavity of the mask or goggles. Skin contusions, exophthalmos, and pain are the sequelae. The Ama of Japan effectively eliminate this problem by attaching small air-filled bulbs to the goggles. As they descend, the pressure gradually collapses the bulbs and the pressure differential is avoided. Breathhold divers who normally wear face masks avoid this problem

CHART A: HIERARCHY OF DIVING RESPONSES



by exhaling via their noses into the mask. This equilibrates the pressure, but materially compromises the diver's air supply in contrast to the system that the Ama use. John Lilly discusses the visual acuity in the porpoise.[88] He observed that dolphins can see in and out of water equally well. This is attributed to two factors. First, the peculiar shape of the cornea permits extreme accommodation. Second, the iris has a U-shaped split. This, Lilly believes, reacts to light and consequently has a different shape for land and water.

Human divers frequently experience difficulty equalizing the pressure differential between the external water pressure and the middle ear cavity. Ear pain, "popping" of ears, tinnitus, blood-tinged sputum, and bleeding from the ear canal during and after diving suggest that the ear drum has been traumatized because of inability to satisfactorily equalize this pressure and thereby "clear the ears." Marked individaul variations in this ability, which probably represent anatomical differences in the middle ear and eustachian tube structures, are observed.

Continued diving is not without hazard to the ear. Harashima and Shigeno observed many more otorhinolaryngological problems in the diving women of Japan than in their non-diving counterparts.[52] In fact, they found it to be the chief occupational disease of the Ama. Problems included chronic otitis media, opacity of the tympanic membranes, stenosis of eustachian tubes, sinusitis and rhinitis. Fifty percent of the Ama in this study demonstrated severe hearing loss. These conditions, generally, showed improvement during the non-diving season. The initial otitis was thought to be a sequela to irritation and occlusion of the eustachian tubes by sea water.

Pressure regulation in the middle ear cavity of aquatic mammals may be a passive phenomenon. Odend'hal and Poulter studied the middle ear cavity in the sea lion.[97] Three significant anatomical differences from the human were noted. First, the ratio of the foot plate to the tympanic membrane was 1:1 as compared to the 20:1 ratio in man. Second, the mucous membrane internal to the tympanic membrane was attached only in the area of the epitympanic recess. The epitympanic recess contains the middle ear ossicles. In other places the membrane was only attached by the veins perforating the temporal bone. Finally, the mucous membrane was composed of three layers. The middle layer of this membrane was a "complex network of venous channels and sinuses imbedded in a matrix of loose connective tissue."[97] These investigators suggested that as a relative negative pressure develops in the middle ear when the external pressure increases with descent, the sinuses passively fill with blood in order to eliminate the pressure differential. The result is distention of the mucosa and eventual obliteration of the middle ear cavity with noncompressible blood. Further descent would not distend the tympanic membrane. The remaining air was compressed into the small epitympanic cavity. This permitted the tympanic membrane and ossicular system to transmit vibration and make hearing possible. The reverse phenomenon would occur with ascent.

The third of the miscellaneous adaptations to be discussed is that of water conservation and balance. In humans, immersion in water is noted to have several effects on fluid volume. The most striking manifestation of this is a diuresis. Immersion initially increases the effective intravascular volume. This is attributed to the hydrostatic pressure gradient forcing the blood from the relatively large venous reservoirs of the lower extremities into the central circulation. McCally feels that the diuresis is directly related to this relative increase in fluid volume.[92] He summarized this by stating, ". . . immersion diuresis is due in part to the inhibition of release of antidiuretic hormones due to distention of cardiac atrial stretch receptors by an associated increase in intrathoracic blood volume produced by the hydrostatic pressure gradients of the immersion medium."[92] Observations of instructor personnel at the Submarine Escape Training Tank, Pearl Harbor, Hawaii, revealed findings consistent with this hypothesis.[12] Forty-five minutes after onset of diving, a diuresis was observed. Four hundred to seven hundred cubic centimeters

of urine were produced in the two and one-half hour work period. This almost exactly paralleled the one and two-tenths to two and one-half pound individual weight losses measured during the diving period. Marked decreases in specific gravities were noted. They ranged from 1.000 to 1.006. Associated findings in this study included threefold elevations in blood urea nitrogen, a marked drop in serum carbon dioxide (CO_2 Paradox), and decreased concentrations of chloride, sodium, potassium, and urea in the urine. Immersion diuresis in this study was not affected by extraneous administration of antidiuretic hormone.[12] Two sequelae to the diving were noted. First, fifty percent of the divers had an unexplainable diaphoresis one to one and one-half hours after completing the dives. The second, was described by Bond as a transient "minor disturbance of motor coordination."[12] This lasted up to eight hours after the dive and was reflected by alteration in motor coordination, changes in penmanship, decreased bowling scores, and interference with golf performance. The etiology and pathophysiology of these changes were not explained, but may be related to this diuresis. These observations are almost diametrically opposed to the water conservation mechanisms of the aquatic mammal. First of all, aquatic mammals that live in sea water must conserve their fluid stores. Water for hydration comes from metabolism of foodstuffs, rather than ingestion of sea water. Second, during the dive, urine flow would be virtually zero, due to the profound vasoconstriction of the renal vasculature. Smith observed that urine production in the fasting harbor seal ranged from 0.06 to 0.1 milliliter per minute while increasing to one milliliter per minute after ingesting a meal of herring.[134] Lowrance, et al., found that apnea and partial anoxia (breathing 10 percent oxygen in nitrogen) produced similar fluid conservation measures.[89] Water and sodium output were decreased due to increased tubular reabsorption of sodium. In contrast, human and canine subjects responded to this partial anoxia by increased urine volume and electrolyte output. Further studies in the seal by Murdaugh, et al., revealed that osmotic diuretics did not increase the urinary output unless they were given with food.[93] However, plasma volume expanders, in these experiments, produced a hypertonic diuresis analogous to the postprandial diuresis. Atropine, which abolishes the bradycardia response to diving, apparently abolished this water conservation measure and suggests the close relationship between the cardiac reflexes, peripheral vasoconstriction, and renal function. Also, it was possible to induce diuresis in the seal by administration of mercurial diuretics and to abolish it by administration of pitressin.[14] These studies indicate that the plasma volume and renal vasculature are instrumental in regulating renal function in the seal. Excretion of metabolic waste products and excess electrolytes occur only when sufficient water is available for urine formation. Vasoconstriction, with resulting decline in renal blood flow and glomerular filtration rate, limit water excretion at other times.

The seal's ability to conserve water, in contrast to the immersion diuresis of the human, is another indication that the extraordinary diving ability of aquatic mammals consists of more than acquired adaptations. The significance of this fluid conservation has not been fully assessed. Undoubtedly, it permits the aquatic mammal to achieve adequate hydration in spite of its hypertonic sea water environment. Second, the virtual absence of renal perfusion and diuresis is consistent with the oxygen conserving reflex and the sustained apnea associated with breathhold diving. Finally, this fluid regulation may be important in prevention of decompression sickness. Further elaboration of this will occur in the conclusion of this paper.

Cold water adaptation and tolerance is the fourth miscellaneous adaptation to be discussed. Immersion in water presents a special challenge to the body's heat conservation mechanisms. The amount of heat required to increase identical quantities of water and air is about 1,000 times greater for water than for air. This is termed the specific heat. In addition, water's thermal conductivity is 25 times as great as air. Beckman summarizes these effects by saying, "The rate at which heat is conducted from the immersed body is

so rapid that heat loss is limited primarily by the rate at which heat is transferred by the blood from the central core of the body to the skin."[10]

Animals exposed to cold environments have developed a number of morphological changes to cope with this stress. These are very apparent in the aquatic mammal, but are also present, in varying degrees, in the human diver. Probably, the most primitive adaptation is poikilothermia, or adjustment of body temperature to that of the environment. This, however, necessitates a decrease in metabolic rate and depression of mental function. Except for minute drops in body temperature, hibernation, and iatrogenic hypothermia, poikilothermia, as a means of protection from low temperatures, is limited to nonmammalian vertebrates. The depressed conscious state and activity level is not consistent with the demands that diving places on the aquatic mammal.

Improved insulative properties of the integument and subcutaneous tissues are important adaptations to counteract the effects of exposure to cold. They achieve what the diving suit attempts to do for the human. In addition, this insulative layer, especially in the behemoth whales, may offer some protection from the tremendous pressures associated with deep dives. Individual, sex and racial differences in the insulative properties of human subcutaneous tissue are apparent. These will be discussed shortly.

Reduction in limb size and vascularity with corresponding decrease in the total surface area to mass ratio is another important morphological change to conserve body heat. The limbs of aquatic mammals consist mainly of fibrocartilage, bone, and skin webbing. The powerful musculature used for propulsion lies primarily in the main body axis of the mammal. These changes, coupled with shunting of blood from the periphery, would essentially eliminate heat loss from the skin, flippers, and flukes, during the dive.

Radiographic studies in humans have demonstrated that the lungs and liver contain more blood when the skin is cold than when the skin is exposed to warm environments.[45] This suggests that the human can make physiological adjustments to cold environments, also. In the human, it has been estimated that 71% of the body is within 2½ centimeters of the surface.[18] Burn chart calculations indicate that approximately 63% of the human body surface area is attributed to the limbs, head and neck.[54] This tremendous area for heat exchange is a marked contrast to the diminution of limb size and streamlined contour of the aquatic mammals.

A fourth consideration in heat conservation is the reduction of heat losses during the exhalation phase of respiration. Beckman reported that 9 to 24% of the total body heat generated is lost during expiration.[10] Apnea during diving and hypoapnea during recovery could substantially reduce body heat losses.

The final consideration on the subject of adaptations to cold water exposure is regulation of basal metabolic rates and reduction of shivering thresholds. Seals are noted to shiver during the recovery phase of strenuous dives. Scholander, Irving, and Grinnell felt that this was due to a general depression of metabolic processes to as much as 50% of resting level during the dive, and then compensation for this depressed activity during the recovery phase.[131] The effects of the revascularization of the limbs which would likely be much cooler than the core body temperature could contribute to this shivering response, also. Behnke and Yaglou noted precipitous falls in the body temperatures of human subjects after removal from cold water.[11] They attributed this to a resumption of peripheral blood flow through the skin and extremities and as a result, the return of much cooler blood to the central circulation. This effect may result in collapse and shock. A diet consisting entirely of protein elevates the basic metabolic rate by 20%.[112] This has been noted in Eskimos and is probably important in seals, whose food intake is almost exclusively fish. The beneficial effects on swimming endurance of a high protein diet as compared to a high carbohydrate diet in rats' ability to swim until exhausted was highly significant (to the 0.01 level) in temperatures of 68° to 82° Fahrenheit (20.0°-28.0°C). Depression of shivering threshold is an important heat conservation method.

Although shivering does increase body heat production five- to seven-fold, the demand it makes on the oxygen stores and cellular metabolites overshadows its beneficial effects.[10] While immersed, the result is greater expenditure of heat than without shivering. This is, in part, due to the tremendous heat losses attributed to the limbs when peripheral vasoconstriction is terminated in order to perfuse the muscles involved in the shivering reflex. Keatinge verified this effect with his observation that core body temperature decreased more with work than at rest in water of the 41° to 59°F range (5°-15°C).[77] The heat generated by the work activity was insufficient to compensate for the heat loss from the extremities secondary to muscle blood flow. Thin subjects cool more rapidly while swimming than while motionless in water at the same temperature.[106] This information has special significance to divers and individuals accidentally exposed to cold water. This will be discussed further in the conclusion of this paper.

Considerable information has been accumulated in regard to human adaptation to cold water. These changes, by and large, are manifestations of the generalized adaptations to cold water discussed previously. First, it is well-established that immersion in water, even if relatively brief, markedly lowers body temperature. In the relatively warm 80° to 90°F (27°-32°C) water of the submarine escape training tanks, Bond reported 2 to 10 degree decreases in body temperature.[12] The average was eight-tenths of one degree Fahrenheit drop in core body temperature per 2½-hour diving session. The average core temperature of the Ama of Japan decrease 3.3°F (1.7°C) in a 45-minute work session in the summer in 72°-79°F (22°-26°C) water, while in the winter they decrease 3.9°F (2.2°C) in 50°-55°F (10°-13°C) water.[75] These effects are somewhat compensated for by alteration in caloric intake, oxygen consumption and basal metabolic rate. The caloric intake among the Ama was approximately 1,000 kilocalories per day greater than that of the non-divers.[75] The Ama have a basal metabolic rate 5% greater than their nondiving counterparts in the summer and a 35% greater rate in the winter.[74] Oxygen consumption paralleled the basal metabolic rate changes and increased 40% during the winter.

The second human adaptation to cold water deals with the insulative properties of the skin and subcutaneous tissues. Beckman reports that tissue insulation on the basis of blood flow and subcutaneous fat thickness may vary fifteenfold in humans.[10] Two factors are important. The first of these is body stature. The short, rotund, endomorphic individual has a smaller total body surface area to body mass ratio than the thin, lean, ectomorphic individual. Thus, women with shorter, more rotund builds may have a natural selective advantage to cold water exposure. The second factor is improved body insulation. Women have three times as much subcutaneous fat as men do.[57] Body core temperatures decline in an approximately inverse linear relationship to subcutaneous fat thickness.[78] This may be the explanation of why females dive in the relatively cold Japanese and Korean waters while men do the diving in the South Sea Isles. Rennie, et al., studied the effect of the subcutaneous fat layer further.[114] These investigators measured maximum body insulation "I", which represents the difference between rectal and skin temperatures divided by the rate of heat loss from the skin, in a variety of subjects. The Korean women studied had significantly greater maximum body insulative capacities than Americans of comparable fat thickness. However, this factor was approximately equal when compared with the nondiving Korean women. The Korean men had a markedly decreased insulative capacity due to decreased subcutaneous fat. It was estimated that a 6% increase in total body fat would have essentially the same effect in protecting an individual from cold water as the female Korean divers have.[111] Channel swimmers have surprisingly small decrements in core temperatures, as compared to the Ama, in spite of swimming in 61°-64°F (16°-18°C) water. Kang, et al., say, "Considering the heavy insulative fat of these swimmers and their high sustained metabolic rate, which was two to three times greater than the Ama's—

their body temperature (maintenance) is understandable."[75] The endomorphic somatotype which characterizes the channel swimmer, no doubt plays an important role in protecting the individual from the effects of cold water.

The third human adaptation is elevation of the shivering threshold. The advantages and disadvantages of shivering were discussed previously. The Korean diving women are able to remain at 82°F (29°C) water for 3 hours without shivering, despite a decline in core temperature of 1.8°F (10°C). This was not possible for nondiving Korean women of comparable fat thickness.[111] Conditioned swimmers have been found to increase metbolic rate without shivering.[18] Skreslet and Aarefjord have defined three states in the process of cold water adjustment in scuba divers.[133] The first, the unacclimatized stage is characterized by an increase in metabolic rate. The second, or intermediate stage shows a decline in core temperature as heat loss is not fully compensated for by metabolism. They believe this is due to habituation of the central nervous system. The acclimatized state is characterized by a constant core temperature, even though increases in metabolism are negligible. This, no doubt, represents conservation of heat by elevation of the shivering threshold and lowered heat transfer between the blood and body surface. The adjustment took approximately 45 days. It is very labile, for 17 days after diving was discontinued, these changes could no longer be demonstrated.

The effects of sudden immersion in cold water are very grave. Immersion in 40°F (4°C) water for one hour is estimated to be fatal fifty percent of the time.[18] Showers in 77°F (25°C) water caused increased ventilation. Respirations could not be voluntarily controlled when the water was 32°F (0°C).[80] Repeated immersion resulted in less respiratory response and indicates adjustment. These findings suggest that involuntary aspiration of water and drowning is a real danger when one is suddenly immersed in near-freezing water. A continuum of response is noted as the body's core temperature is depressed.[10] Amnesia occurs at 94°F (34°C) with areflexia and unconsciousness at 86°F (30°C). At 90°F (32°C) arrhythmias occur. Ventricular fibrillation is the usual terminal event in death from cold water exposure. Initially, it may be due to an adrenergic response and hyperventilation. The electrocardiogram in hypothermia is characterized by slow rate, low voltage, inverted "T" waves, elongated "QRS" complexes and appearance of the abnormal "S" wave. The ideal treatment for cold water exposure is immersion in water of 100°-105°F (38°-41°C). Behnke and Yaglou reported that in response to this treatment, skin temperatures rose abruptly while the core temperatures continued to fall.[11] The rate of temperature decrease was greater than during immersion and suggests that the vasoconstrictive response was obliterated and the peripheral circulation reconstituted. However, with body temperatures below 86°F (30°C) external warming may be of no value, because there is no effective circulation. In this situation, cardiopulmonary bypass has been used successfully in reviving victims of profound hypothermia.[31]

In summary, it is apparent that the human's ability to tolerate cold water is very meager when compared to the aquatic mammal. Yet, virtually all the compensating factors observed in the aquatic mammal can be demonstrated to some degree in the human breath-hold diver.

The final topic of the miscellaneous adaptations to be discussed is that of navigation. The ability of porpoises and closely related species of aquatic mammals to navigate underwater has been investigated considerably by Kellog and Lilly.[81, 88] The so-called "dolphin language" and "superhuman" brain are manifestations of this ability. While swimming, porpoises emit low frequency sounds. These are reflected off obstacles and then discriminated in the massive cortical areas of their brains. The concept is identical to that of sonar. Porpoises have extraordinary ability to discriminate and integrate this information. They can distinguish edible from non-edible fish of identical size on the basis of this sonar, as well as travel through obstacle courses at near maximum speeds without difficulty. No counterparts of this sys-

tem have been described in the seal and sea lion family.

C. PROPULSION IN THE AQUATIC ENVIRONMENT

The third major portion of this thesis deals with underwater swimming and propulsion in the aquatic environment. Again, the efforts of human swimmers and breathhold divers can hardly be compared with those of the aquatic mammal. In terms of forward propulsion, seals primarily employ their pectoral fins, while the porpoises use an oscillating movement of their flukes. The porpoise is the more proficient swimmer. Kellog has stated that porpoises are capable of swimming continuously at twenty knots, and reports of these mammals swimming alongside ships cruising at thirty knots are not infrequent.[81] However, recent studies indicate that their actual top swimming speed may not be nearly this great. Lang and Norris of the Naval Ordnance Test Station in Pasadena, California, recorded swimming speeds of porpoises, by using motion picture cameras, stop watches, and accurately calibrated boat speedometers.[140] The porpoise's maximum swimming speed was sixteen and one-tenth knots in calm water, and fourteen and five-tenths knots in rough water. Initial studies of propulsion in porpoises were plagued by the discrepancy between the theoretical speeds calculated (on the basis of muscle mass, fin and fluke movement, and displacement) and the observed swimming speeds.[81] The observed speeds were much greater than the theoretical speeds. Three explanations account for this. First, the porpoise has a very streamlined and efficient hull form in terms of hydrodynamics. The blunt, round contour reduces turbulence and drag, while traveling at rapid speeds. Second, the subcutaneous layer of fatty tissue is of an oily consistency. In addition to providing excellent insulation against the cold water, it tends to assume the contour of the water flow pattern and reduce drag. The rostral end of the porpoise's skin, where smooth water flow occurs while swimming at high speeds, is nearly bloodless. The subcutaneous tissue in the area adjacent to the flukes, where turbulence and drag normally appear, is very vascular. It has been hypothesized that this increased vascularity would assist in producing a laminar flow pattern by decreasing turbulence through heat transfer.[140] Finally, porpoises appear to be adroit "surfers." It is known that as a boat advances through the water an advancing pressure field is generated by the bow. Porpoises apparently utilize this pressure field while swimming in front of ships. It may be an explanation for the observation of porpoises traveling alongside ships at thirty knots speed.[141]

Analysis of swimming times in world caliber competitors reveals that these swimmers are capable of swimming for short intervals at 4½ knots, and for long distances at 3½ knots. Adrian, Singh, and Karpovich reported that energy costs of the kick, corresponding somewhat to the fluke movements of the porpoise, are to to four times as great as the arm stroke which corresponds to the flipper movements of seals.[1] The efficiency of the leg stroke varied from 0.05% to 1.23, while that of the arms varied from 0.56 to 6.92% Oxygen consumptions for 15 meter swims at 1 meter per second were 16 liters per minute for the legs and 4 liters per minute for the arms.[1] There are three explanations for these findings. First, the 16 liters per minute oxygen consumption is a theoretical calculation based on a brief time interval. Second, the muscles of the hips and lower extremities are some of the largest in the body and have correspondingly high oxygen demands. Third, the range of efficient propelling movements of the legs is relatively small when compared to the highly mobile arms. Consequently, when unassisted, the legs are much less efficient than the arms as a means of propulsion. Observations of long distance swimmers suggest increasing emphasis on the arm stroke and reducing the kick to slow efficient movements designed to assist in stability in the water. Swimming fins do, however, counteract this effect, for efficiencies of finned swimmers have been reported to vary from 2 to 8%. This corresponds favorably to the efficiency of the arm stroke. Yet, fin swimming is only 20% as

efficient as walking or running on dry land.[86]

Studies of underwater swimming has shown that an average swimming rate of seven-tenths to nine-tenths knots is most efficient.[49] At speeds greater than eight-tenths knots, efficiency declines progressively, based on oxygen consumption. Marked variations were observed with training, body build, and water temperature. Specht, et al., found that swimmers with the lowest kick rates had the highest efficiencies in swimming.[136] They suggested that ease of vertical position control, that is to say, neutral buoyancy, was the significant factor.

Surprisingly, maximum respiratory responses of underwater swimmers using modified scuba gear were as much as 50% less than the responses while using this equipment in the air.[136] Increasing oxygen debt, carbon dioxide retention, and fatigue are the expected results. Studies of McArdle on oxygen consumption of swimming rats support this information.[91] Weights were attached to the rats. This made swimming on the surface increasingly difficult. Decreased aerobic metabolism and decreased swimming efficiency resulted. Eventually, swimming movements ceased, and drowning occurred. This sequence of events may account for some of the deaths attributed to scuba diving.

With this information in mind, the diving patterns of the Ama are particularly significant. The Cachido, or shallow water divers, descend to fifteen feet unassisted. They dive for 30 seconds, and rest for 30 seconds. The Funado, or deep divers, descend to 60 feet with weights and are pulled to the surface by their assistants. Descent and ascent account for 15 seconds each. About 30 seconds is spent on the bottom. The surface intervals of these divers are usually 60 seconds. Thus, the energy expenditures and oxygen consumption for each of the types of divers are approximately equal. Craig and Medo found that in repetitive diving, the excess volume of oxygen required was no more than the sum of the oxygen required for single dives.[27] They estimated that the breathhold diver, in their experiments, could work at half maximal capacity for 30 seconds, without lowering pulmonary oxygen levels to the danger point. This corresponds well to the Ama's diving patterns. Additional practical and theoretical application of this analysis of propulsion in the aquatic environment will be discussed subsequently.

CONCLUSIONS

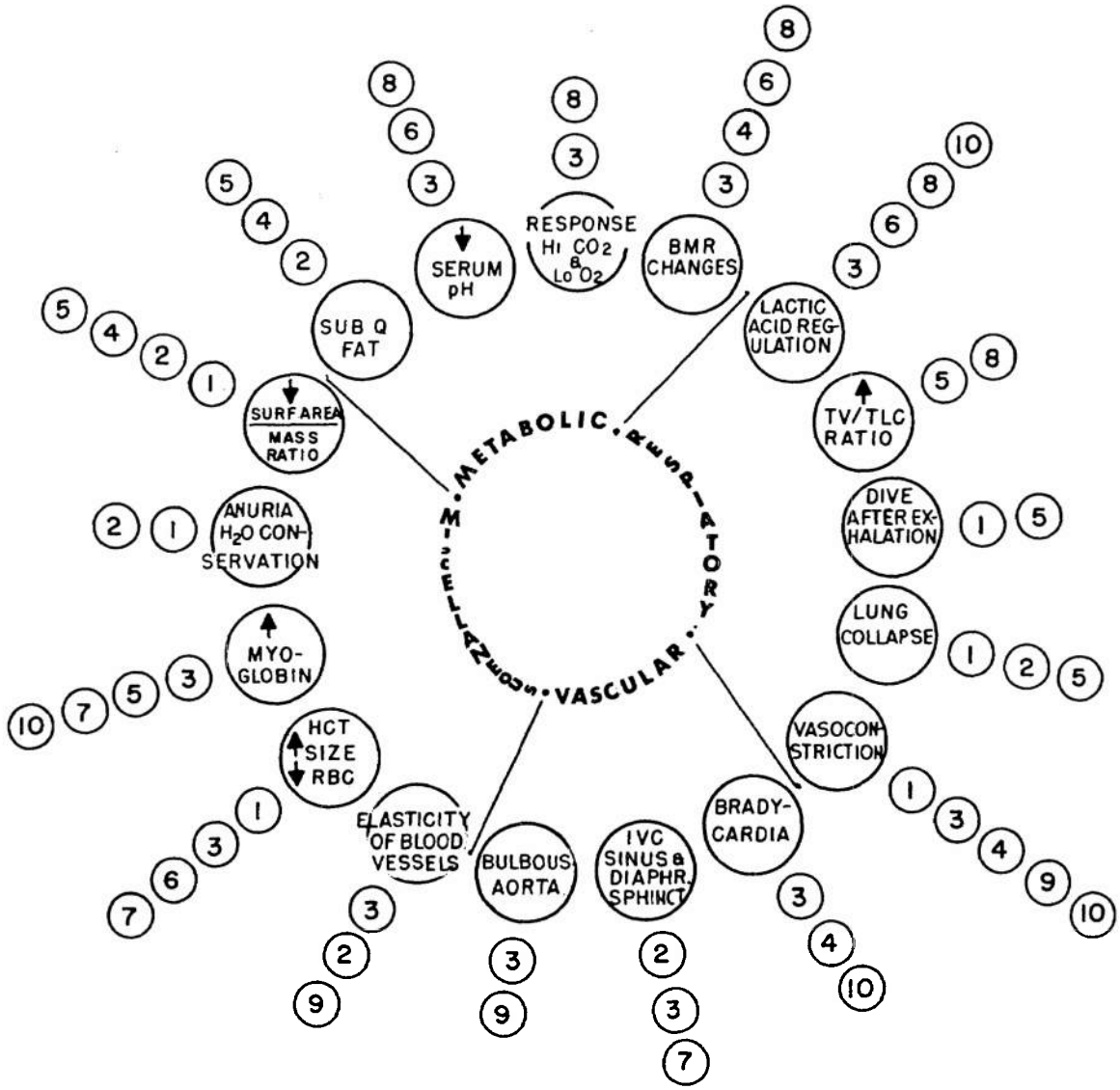
When the previously discussed adaptations to breathhold diving are viewed in toto, several conclusions are noted. First, virtually every aspect of diving is replete with unanswered questions. In many cases the research was performed on a single representative of the aquatic mammals. Conclusions were often based on single observations, rather than carefully controlled investigations. Conflicting data were all too common. Second, the adaptive responses of the so-called "ideal" aquatic mammals show a remarkable degree of integration. For example, as body shape has become rounded, the surface area of the limbs has been greatly reduced. This not only is important in conserving body heat, but it also increases the animal's tolerance to pressure and improves its hydrodynamic contour in regard to propulsion. Peripheral vasoconstriction, both arterial and venous, during breathhold dives, not only permits perfusion of the critical organs, but it has important corollaries to cold water adaptation, anaerobic metabolism of muscles, fluid conservation, prevention of thoracic squeeze, increased oxygen utilization from the blood, and protection against nitrogen narcosis. Diving after full expiration may assist in descent due to decreased buoyancy. More important, however, is its effect in prevention of nitrogen narcosis, decompression sickness, and fluid overload of the heart. Initiation of the inferior venal caval sphincter mechanism and regulation of dive durations could also be related to this phenomenon. The bradycardia and peripheral vasoconstriction responses conserve core body temperature, decrease cardiac oxygen demands, and permit adequate perfusion of the brain. The anatomy of the respiratory tract is not only highly efficient for exchanging large volumes of air with minimal respiratory effort and conserving heat, but it is

so constructed to permit lung collapse and thus minimize the possibilities of nitrogen narcosis during deep dives, and decompression sickness after surfacing. The subcutaneous fat and oily tissue is not only important for protection from the cold, but it improves the aquatic mammal's ability to propel itself in the aquatic environment. The highly elastic walls of the arterial vasculature insure maintenance of the mean blood pressure during bradycardia and continuous perfusion of the brain, but are probably also important in achieving effective vasoconstriction and tolerating the demands that increased ambient pressure make on the cardiovascular system. Elevation in hematocrit and decrease in red blood cell size improve the oxygen transport and storage abilities of the blood. The decrease in erythrocyte size may offer some degree of resistance to the effects of vascular stasis, and protect the animal from decompression sickness. Further elaboration of this will occur shortly. Thus, except for perhaps the visual and navigational adaptations discussed, virtually every adaptation is related to several body systems, is multifunctional, and is integrated into the general diving response. Chart B is a schematic diagram of the adaptations associated with breathhold diving, their relationship to each other, and their integration into the general diving response. Third, review of this material suggests new interpretations and possible solutions to problems that plague the human exposed to increased ambient pressures and the aquatic environment. Some of the relationships are only of a coincidental nature, some now have direct practical application, and others are only of a theoretical nature at this time.

Several examples of coincidental developments, that is to say, problems solved by man in regard to diving and well-established in the aquatic mammal, are sonar, submarine hull shape, and development of the wet suit for cold water exposure. The discriminatory ability of the porpoise's "sonar" system is very astute. As neurophysiological studies further delineate this remarkable ability in porpoises, keys to improving the sonar system for navigation may be realized. In regard to understanding the effects of hull form and propulsion in the aquatic environment, one wonders if it is more than coincidence that at approximately the same time that the hydrodynamic superiority of the porpoise's whale's body configuration was recognized, submarines assumed a radically new contour. For submarines, the sharp, knifelike configuration of the destroyer hull was changed to the blunt, whalelike shape seen in the modern underwater boat. Information from the study of porpoises suggests several other areas of investigation in terms of propulsion. First, a heat exchange system with a partially compressible external hull near the stern may significantly reduce drag and noise from water turbulence and increase underwater speed, as research on the porpoise has indicated. Second, rather than use a rotary propeller for small two to three man underwater vehicles, an oscillating tail assembly system similar to the porpoise's fluke movements might be considered. The advantages of increased speed and decreased noise production, especially for reconnaissance purposes, might be realized. The wet suit used by human divers attempts to achieve what the subcutaneous fatty tissue layer does achieve for the diving mammal. Inquiries into finding a material, perhaps a semifluid, with the insulative abilities of fatty tissue, is indicated. Certainly, if it were of a noncompressible substance in contrast to the "foam" suit used today, the range of depths for which the suit is useful would be much improved. Experiments with extrinsic heat sources for warming diving suits, in the form of chemical, electrical, or atomic energy are being investigated. The potential dangers, expense, and cumbersomeness of the present systems restrict their use.

There are several practical applications important to the human diver that a review of this material suggests. First, in response to cold water exposure, struggling is contraindicated. It will counteract the body's own defense of shunting blood away from the periphery in order to preserve core temperature, increase heat losses secondary to tachypnea, and minimize the protective ef-

CHART B: INTEGRATION OF THE DIVING RESPONSE



Key: Numbers in Circles indicate that the responses contribute to the factor denoted in the indicated section of the key

I. PROTECTION FROM	II. IMPROVED EFFICIENCY IN	III. MAINTENANCE OF
(1) Bends	5 (5) Propulsion & Buoyancy	9 (9) Cerebral Perfusion
(2) Squeezes	6 (6) Oxygen Usage	10 (10) Anaerobic Metabolism
(3) Anoxia	7 (7) Oxygen Storage	
(4) Cold Water	8 (8) Recovery	

fects of the victim's clothing by promoting circulation of water. The second practical application is in regard to underwater propulsion. Ideal swimming speeds, body positioning, and leg motion have been determined for greatest underwater swimming performance. The physiological changes and improved performance of repeated practice in breathhold diving is well documented. Effects and dangers of fatigue, hypothermia, and equipment limitations are also realized from this study. In terms of energy costs, research into reducing the leg movement and increasing the arm movement in swimming underwater should be considered. Such unique ideas as translating the arm movements to a propeller shaft, traveling in a semirigid porpoise-shaped vehicle in order to reduce drag, or perhaps use of the legs to provide energy to move an oscillating, fluke-like appendage, are mentioned as possibilities for research which could improve underwater swimming performance. The third practical application of this research deals with exploitation of the aquatic mammal. Porpoises, seals, and whales are highly trainable, as performances at marine shows verify. Porpoises in the United States Navy Sea Lab II Project were utilized for rescue and messenger purposes. They are to be used in the Sea Lab III Project, also. It seems feasible that these mammals could be trained to act as lifeguards, locate sunken ships, protect human surf-bathers from shark attacks, herd fish into nets, harvest edible sea foods, and participate in underwater reconnaissance and submarine detection work. The porpoise's learning capacity, swimming and diving ability, and underwater navigation system (sonar) lend themselves well to these skills.

The final group of applications are theoretical. Their ultimate role in diving, especially in regard to humans, has not been determined. The first of these deals with the prevention, understanding, and treatment of medical problems of diving, namely nitrogen narcosis, thoracic squeeze and decompression sickness. In terms of prevention of these diving medical problems, one need only to look at the respiratory adaptations demonstrated in the seal for clues. First of all, oxygen stores are primarily those dissolved in the blood, a liquid and correspondingly noncompressible substance. The compression of the air-filled lungs, which normally occurs with descent, is compensated for by distention of venous sinuses with blood. The answer for the human diver, who does not have this natural adaptation, is to breathe a fluid medium in which oxygen is dissolved. Ventilation would be independent of oxygen content, depth, or rate of ascent. Thoracic squeeze, nitrogen narcosis, and decompression sickness would thus be prevented. Unfortunately, the problems of carbon dioxide elimination, aspiration of fluid from the lungs, and increased airway resistance have not been solved as Kylstra and others have demonstrated in experimental animals.[85] Fluid respiration could be the ultimate answer for deep human dives, free ascent submarine escapes, and prolonged diving. The solution may lie in finding an "ideal" oxygen transport medium. That is to say, one that is physiological, has a lower viscosity than water, has high affinity for carbon dioxide, and readily releases oxygen.

In terms of understanding the pathophysiology and treatment of decompression sickness, perhaps closer attention should be given to fluid balance. As noted earlier, immersion of humans in water promotes a diuresis, whereas the opposite response is evoked in seals. Measurable fluid volume losses have been demonstrated in humans after immersion.[12, 142] Experiments by Cockett, et al., have shown that animals who were given rapid intravenous infusions of blood and fluids tolerated an explosive decompression schedule that proved to be lethal to a similar group of untreated animals.[19, 20, 21] Thus, the role of adequate hydration and intravascular fluid volume in prevention of decompression sickness may be more significant than previously realized. This concept is consistent with the increased bends susceptibility of individauls with injuries, post-alcoholic "hangovers," and systemic illnesses. A tentative explanation for these findings is offered by Heimbecker and his associates.[56] The microcirculation of the dog conjunctiva and the cheek pouch of the golden hamster were

studied during hyperbaric conditions and rapid decompression. Of the animals that died from the decompression, "... all showed large gas emboli, producing marked embolic obstruction throughout all parts of the vascular tree."[56] The microcirculation in those animals that survived the insult revealed, "... a marked reduction of tissue perfusion with capillary slowing and stasis, intravascular hemoconcentration, and red cell agglutination; tissue edema; variable arterial and arteriolar constriction..."[56] This reached a peak fifty minutes after decompression and slowly improved over the next hour. The authors emphasize that no gas embolism was observed in the viable animals. These investigators concluded that decompression sickness is due to inadequate tissue perfusion and gas embolism occurs only as a terminal event. Philip's study, which demonstrated that heparin and the lipemic clearing agent, depolymerized hyalurenate, ameliorated the effect of decompression sickness, whereas coumadin with its purely anticoagulant effects did not, lends further support to this pathophysiological mechanism for understanding decompression sickness.[105] The diminution in the size of the seal's red blood cell may be a factor in tolerating the stasis and anoxia associated with the peripheral vasoconstriction response. This may afford some degree of resistance to decompression sickness. Chart C is a stepwise summary of the proposed mechanism and the indicated treatment for decompression sickness in light of these investigations. Perhaps heparin and fluid therapy are indicated during decompression from exceptional and extreme dives.

The second theoretical application of this research deals with direct applications to clinical medicine. The effects of strenuous diving on humans is just being realized. Aside from damage to hearing, the effects of diving to moderate depths appear to be salubrious. Harashima and Shigeno report that the Ama of Japan have much lower rates of central nervous system vascular diseases than a group of control Japanese.[52] The psychological, physiological, and metabolic effects of physical conditioning have not been fully determined, but appear to be conducive to good health. The beneficial effects of immersion in physical therapy have been long known. The Jobst Stocking, useful for treating vascular diseases of the legs, was a direct application of the beneficial effects of the pressure gradient on removing excess fluid from the lower extremities. Immersion may have value for its diuretic effect, also. Another line of inquiry deals with the investigation of shock. In many respects, the physiological changes associated with breathhold diving are analogous to the responses associated with the shock syndrome. It might be fruitful to investigate this relationship further. When does the diving reflex become irreversible? Does the diving mammal have special metabolites to prevent this response from progressing into an irreversible process? What stimuli cause the aquatic mammal to surface and so terminate the diving reflex? The relationship between surgical hypothermia, peripheral vasoconstriction, adaptation to cold water, and the diving reflex is apparent. Perhaps these could be utilized in surgery for reducing exsanguination and decrease the possibility of brain damage occurring during anesthesia. The proposed mechanisms for prevention and treatment of decompression sickness may have some direct applications to problems found in the peripheral vascular diseases. In dealing with respiratory diseases, slowing of respiratory rate is known to improve oxygen extraction in the lungs. This principle is employed in the treatment of obstructive airway diseases and is remarkably similar to the ventilatory pattern of the aquatic mammal and the well-conditioned breathhold diver.

The final theoretical application which research of this variety may have is in selection of diving personnel and prediction of diving performance. For example, requirements for Navy divers are based on personal motivation, physical fitness, ability to withstand claustrophobia, tolerance of 100% oxygen at 2 atmospheres pressure, and ability to clear one's ears. These criteria are important, but they do not actually predict the individual's performance as a diver. Primarily, they reveal whether or not the diver can tolerate the physical demands of the diving training pro-

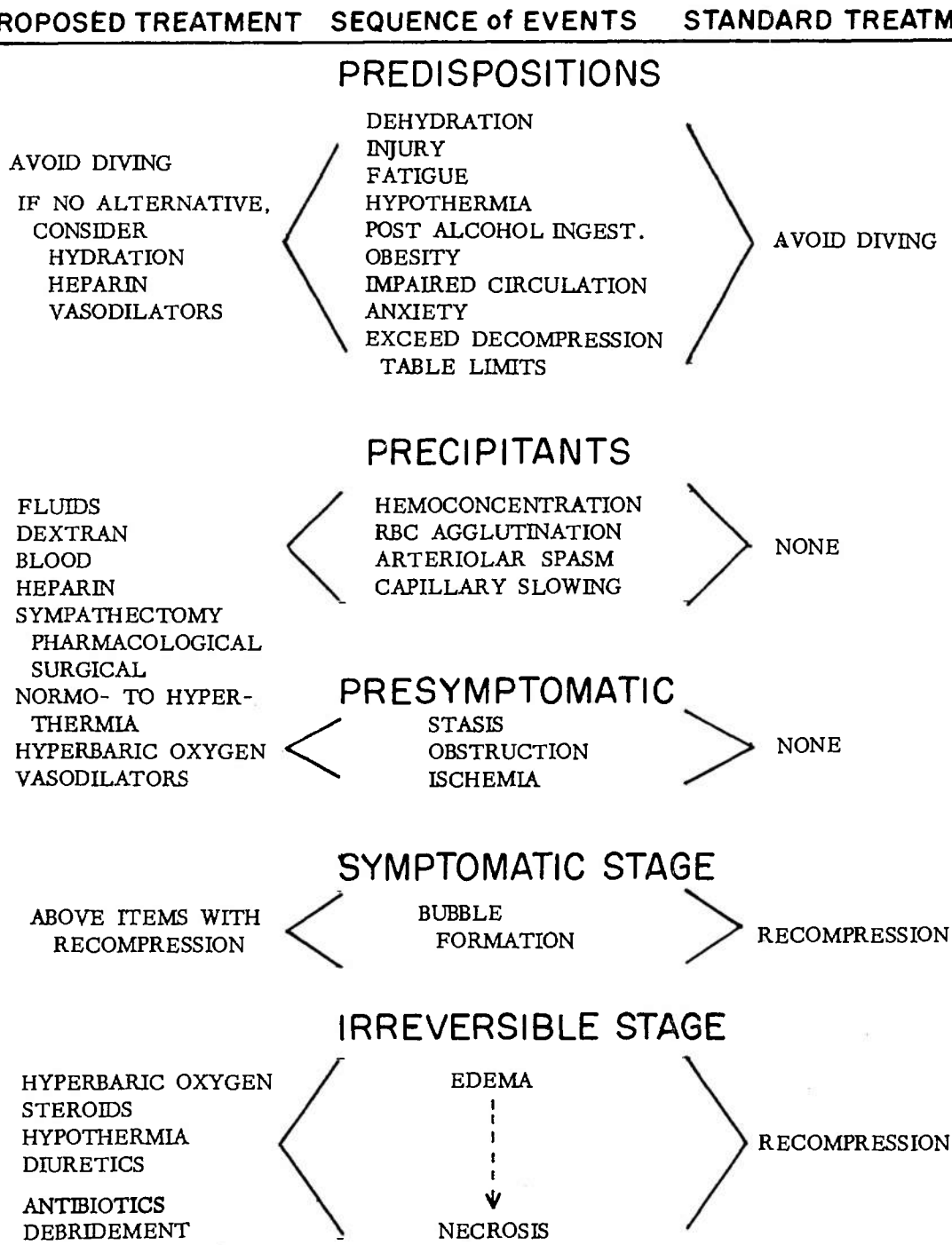
TABLE V: MAN VS. THE "IDEAL" DIVER

CONDITION	ADAPTATION SEEN IN THE "IDEAL" DIVER	NATURAL ADAPTATIONS IN MAN	COMPENSATIONS HUMANS USE AT PRESENT	THEORETICAL COMPENSATIONS
	**********CARDIOVASCULAR PROBLEMS**********			
ARRHYTHMIAS	Absent	Selection*	Limit Diving	? Drugs
O_2 USAGE	Acidosis	Acidosis	Conditioning	?
O_2 STORAGE	Myoglobin, Size RBC	?	Conditioning	?
	**********COLD EXPOSURE**********			
BODY FORM	Blunt, Rounded	Selection*	Wet Suit	Heated Suit
INSULATION	Sub Q Tissue	Selection*	Wet Suit	Improved Insul.
LIMB VASCUL.	Decreased	Selection*	None	? Drugs
REGULATE BMR	Present	?	Exposure, Diet	? Drugs
ELEVATE SHIV. THRESHOLD	?	Selection*	Conditioning	? Drugs
	**********FLUID REGULATION**********			
WATER CONSV.	Anuria	None	None	Drugs, Fluids
	**********NAVIGATION PROBLEMS**********			
UNDERWATER TRANSIT	"Sonar"	None	Compass	Subminiature Radio, Sonar
	**********OPHTHALMOLOGICAL PROBLEMS**********			
UNDERWATER VISION	"U" Shaped Cornea, Extrem. Accomodation	None	Mask	? Contact Lens Miotic Agents
CORNEAL IRRITATION	?	None	Mask	?
	**********OTOLOGICAL PROBLEMS**********			
EAR SQUEEZE	Venous Sinuses	None	Drugs, Surg.	?
	**********PROPULSION**********			
CONTOUR	Hydrodynamically "ideal"	Buoyancy, Kick, Effic.	Conditioning	Hull Forms
MOVEMENT	Fin or Fluke	Arms & Legs	Swim Fins	Aquamobiles**
	**********RESPIRATORY PROBLEMS**********			
SQUEEZE	IVC Sinuses	Increase TLC	Scuba	Fluid Resp.
BENDS	Exhalation, Collapse Lungs	None	Decompression, Mixed Cases	Fluid Resp.
N_2 NARCOSIS	Exhalation	None	Mixed Cases	Fluid Resp.
BREATHING EFFICIEN.	TV---)TLC	Selection*	Conditioning	?
CO_2 TOLERANCE	?	None	Conditioning, CO_2 Absorbers	Fluid Resp.

*i.e. Natural selection of divers most resistent to these problems

**Underwater vehicle that will convert arm and/or leg movement to a more efficient underwater propelling mechanism

Chart C: PROPOSED ETIOLOGY, PATHOPHYSIOLOGY AND TREATMENT FOR DECOMPRESSION SICKNESS



gram. Criteria such as the bradycardia response to immersion and cold water exposure have been suggested as prerequisites for the successful diver.[47] The tissue insulative factor, as described by Rennie in his study of tolerance to cold water, is another consideration.[113] Swimming ability, while not important per se, is related to this discussion. The beneficial effects of buoyancy and leg kick efficiency have been described previously. Also, the time interval for the pulse rate to return to resting levels after a vigorous swim may provide a measure of the individual's physical condition.[145] These may offer some absolute criteria for selection of divers. Finally, factors such as coagulability of the blood, serum lipid concentrations, shivering threshold, respiratory quotients, composition of diet, relationship between total lung capacities and residual lung volumes, and vascular resistance to ischemia may have value in prediction of a diver's performance and resistance to medical problems associated with diving.

This concludes the discussion of the subject, "Mammalian Adaptations to Diving." The question which was raised initially as to man's role in the aquatic environment is again posed. This thesis suggests that human adaptations to breathhold diving are indeed feeble when compared to those of aquatic mammal. The responses in the human, in fact, represent a general adaptation to stress and conditioning, rather than to the aquatic environment itself. Table V is a comparison of the aquatic adaptations of the "ideal" diver, man, the present methods man uses to compensate for his limited diving ability, and finally theoretical methods for compensating for these deficiencies. However, as has been shown, the human is not totally foreign to the aquatic environment. As knowledge of breathhold diving, aquatic mammals, and underwater physiology, accumulates, it is hoped that the human will approach the diving abilities of the "ideal" aquatic mammal.

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13. ABSTRACT

This thesis is a comprehensive review of the literature dealing with mammalian adaptations to diving. Respiratory and cardiovascular changes are particularly emphasized. Other subjects pertaining to diving adaptations such as fluid conservation, temperature maintenance, propulsion, underwater vision, and navigation are also included in the discussion.

This review suggests that there is a remarkable integration of the adaptations and acclimatizations involved in the breath-hold diving response. On the basis of these studies, a new pathophysiological mechanism for understanding the etiology of decompression sickness is presented in the conclusion. In addition, applications of responses associated with diving to clinical medicine and prediction of diving performance are also discussed. Wherever possible, comparison of human breath-hold divers to aquatic mammals is made.

14. KEY WORDS	LINK A		LINK B		LINK C	
	ROLE	WT	ROLE	WT	ROLE	WT
Adaptations to diving						
Decompression sickness						
Propulsion underwater						
Excursion diving						
Breath-holding						